Review Article

Postoperative healing and complications based on anterior cruciate ligament reconstruction graft type

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Contributions: (I) Conception and design: All authors; (II) Administrative support: All authors; (III) Provision of study materials or patients: All authors; (IV) Collection and assembly of data: None; (V) Data analysis and interpretation: None; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

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Abstract: Injury to the anterior cruciate ligament (ACL) is a devastating injury to athletes of all ages. The current gold standard treatment following complete rupture of the ACL is reconstruction of the torn ligament with autograft or allograft tendon. Commonly used tendon grafts include patellar tendon, hamstring tendon, and quadriceps tendon. Although ligaments and tendons have similar collagen and proteoglycan compositions, they maintain a unique composition and arrangement of cells to serve their unique biomechanical needs. Therefore, following ACL reconstruction (ACLR), the implanted tendon tissue undergoes a process of remodeling which is termed "ligamentization". The process of ligamentization is divided into three main phases, which include the early healing phase, the proliferative phase, and the maturation phase. Following the process of ligamentization, the graft tissue closely mimics the appearance of ligament tissue on an ultrastructural level. Successful outcome following ACLR is contingent upon adequate remodeling of the tissue as well as healing of the graft within the bone tunnels in the femur and tibia. Choice of graft has individual implications regarding their associated risk of complications, failure, and infection. The purpose of this review is to summarize the process of ligamentization and graft healing and to discuss how graft type influences the rate and types of complications, failures, and infections.

Keywords: Anterior cruciate ligament (ACL); reconstruction; graft healing; complications; ligamentization

Received: 22 January 2024; Accepted: 31 May 2024; Published online: 28 June 2024.
doi: 10.21037/aoj-24-3
View this article at: https://dx.doi.org/10.21037/aoj-24-3

Introduction

The anterior cruciate ligament (ACL) is the most commonly injured ligament in the knee, with an annual reported incidence in the United States (US) of 1 in 3,500 people (1). Following an ACL rupture, there is limited potential for healing due to the local environment within the knee (2). Despite a recent resurgence in ACL repairs being performed globally, the current gold standard treatment for an ACL rupture is to reconstruct the torn ligament using either autograft or allograft tissue in patients with instability and those returning to pivoting sports. In 2017 it was estimated that approximately 400,000 ACL reconstructions (ACLRs) were performed annually in the US, although this number has likely increased each subsequent year (1).

Common autografts include bone-patellar tendon-bone (BTB), hamstring (gracilis and/or semitendinosus), and quadriceps (with or without patellar bone). Multiple allografts are available including BTB, hamstring (semitendinosus), tibialis posterior, tibialis anterior, and Achilles tendon (with or without bone). Graft fixation is usually performed within the bone tunnels or on the adjacent cortex with a button or a post-washer device.

Tendons and ligaments share a similar composition of dense connective tissue composed primarily of type I collagen (90%) and type III collagen (10%), proteoglycans,

and cells (3). However, tendons and ligaments also have unique properties required for the biomechanical stresses to which they are subjected. This is demonstrated by the precise composition and arrangement of their matrix molecules (4). On an ultrastructural basis, ligaments contain cells that are more metabolically active, with rounded nuclei and higher DNA content, contain more type III collagen, more proteoglycans, less total collagen, a different amount of nonreducible collagen cross-links, and a different distribution of collagen fibril diameters compared to tendons (5,6).

ACL rupture is a devastating injury to athletes of all levels. Following ACL rupture, only 50–65% of recreational athletes are able to return to the preinjury level of sport (7,8). For professional athletes, it was recently reported that amongst National Football League (NFL) athletes sustaining an ACL injury during their career, only 55.8% were able to return to play postinjury, and of these athletes, only 28.5% remained in the league for at least 3 years postinjury (9). Whether an athlete is able to successfully return to their preinjury level of play is significantly influenced by the ability of the reconstructed graft tissue to remodel and heal; a process termed ligamentization. It is often debated the ways in which graft selection and fixation may influence healing, complications, and failure rate following ACLR.

Therefore, the purpose of this review is to summarize the process of ligamentization and graft healing and to discuss how graft type influences the rate and types of complications, failures, and infections.

Ligamentization

In 1986, Amiel *et al.* (10) used a rabbit model to demonstrate that a tendon autograft used to replace an excised ACL goes through a continuous process of tissue transformation resulting in the transplanted tendon becoming composed of tissue very similar to that of a normal ACL. This process was termed "ligamentization". Since then, this process has been studied in a variety of animal models (10-14).

The process of ligamentization has been categorized into three phases: early healing, proliferation, and maturation (10,15). During the early healing phase, an accumulation of host inflammatory cells, including neutrophils and macrophages (16), are recruited to the periphery of the graft and release various cytokines, such as interleukin-1 (IL-1), tumor necrosis factor-alpha (TNF-α), transforming growth factor-beta (TGF-β), and matrix-metalloproteinase-1,13 (MMP-1,13) (17,18). Ultimately, this increase in cell signaling leads to the digestion of collagen and infiltration of repopulating cells. At the same time, during the early healing phase the tendon graft undergoes avascular necrosis, most notably in its central portion (19). During the process of avascular necrosis, several cytokines are released and lead to a subsequent cascade of growth factors that guide future steps (16,20).

After the graft undergoes avascular necrosis, the graft enters the proliferation phase. This is highlighted by an increased expression of vascular endothelial growth factor (VEGF) leading to vascular ingrowth of the graft. It is theorized that new blood vessels originate from the synovium, infrapatellar fat pad, and the pseudo-ligamentum mucosum (4). Adequate revascularization allows the graft to be infiltrated with new cells and undergo matrix remodeling, which is necessary for successful graft healing (11,21).

Finally, the graft enters the maturation phase. As the name suggests, this phase is characterized by continuous remodeling and maturation of the newly infiltrated cells into cells that closely resemble that of an intact ACL (22).

Based on evidence from animal studies, the time frame for these phases has been defined as the early healing occurring from the time of graft implantation through the 4th postoperative week, the proliferative phase occurring from postoperative week 4–10, and the maturation phase occurring as a continuous process beyond 10 weeks postoperatively, acquiring similar morphology and histologic appearance by 6 to 12 months (22-26), The results of these animals studies also suggest that biomechanical strength of the graft tissue varies greatly throughout the process of ligamentization, with a sharp decline in strength occurring during the early healing and proliferation phase, followed by a gradual return in strength seen throughout the maturation phase (*Figure 1*) (27).

Graft biopsy

The relatively scarce data on graft healing in humans highlight substantial differences in the timeline of these phases in humans. Rougraff *et al.* (28) obtained graft biopsies in 23 subjects with previous ACLR using patellar tendon autografts. Biopsy was performed between 3 weeks to 6.5 years postoperatively. Histologic analysis of the biopsy specimens revealed the early healing, or "repopulation" phase occurred over the first 2 months postoperatively, with one biopsy obtained at 3 weeks postoperatively displaying a viable graft evidenced by increasing fibroblast number and active nuclear morphology. Interestingly,

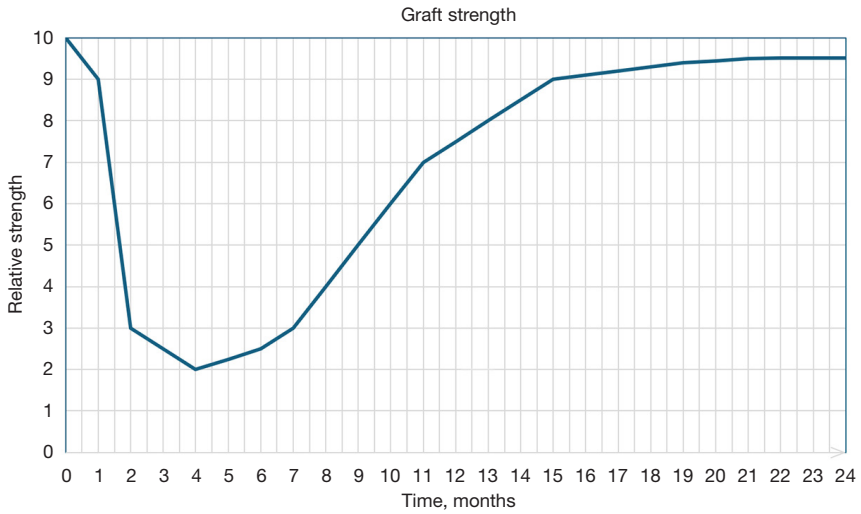

Figure 1 Graphical depiction of mechanical strength changes during the different phases of ligamentization. As shown, there is a sharp decrease in strength during the months corresponding with the early healing into the proliferation phase, with a gradual return in strength during the months corresponding with the maturation phase.

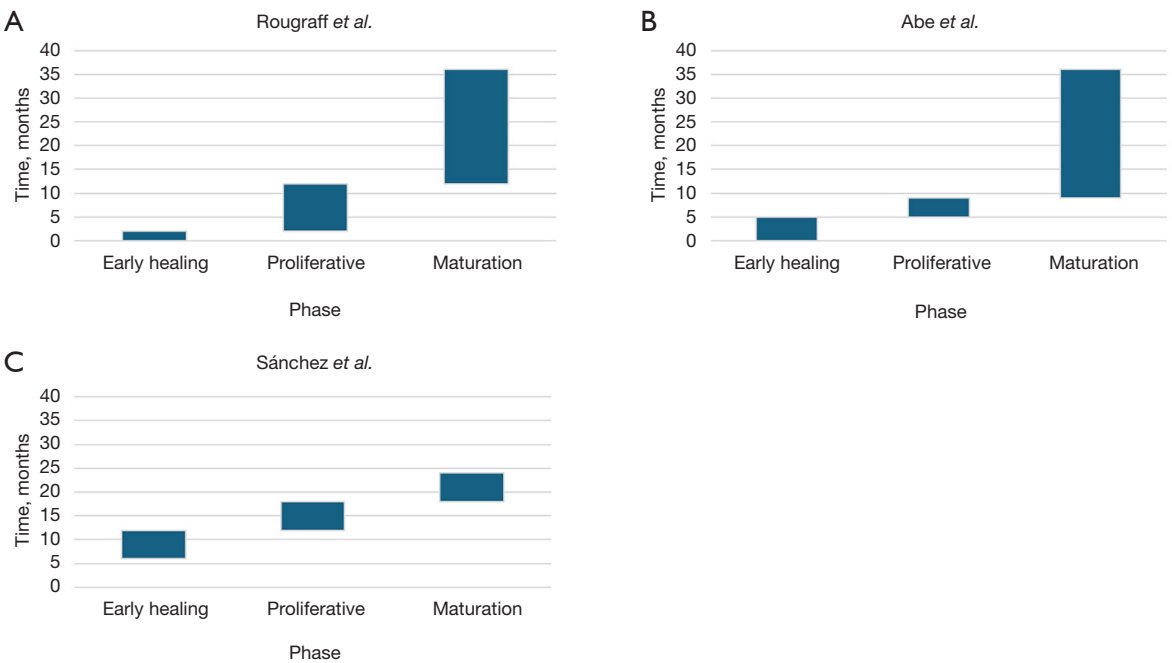

Figure 2 Illustration depicting the timing of each phase of ligamentization based on biopsy data from (A) Rougraff *et al.* (28), (B) Abe *et al.* (29), and (C) Sánchez *et al.* (30).

they did not find evidence of complete graft necrosis during this stage as previously described in animal studies. The process of proliferation then occurred over the next 10 months. During this phase, they noted a marked increase in the number of fibroblasts, active nuclear morphology, and neovascularity. Additionally, there were more areas of degeneration as the percentage of mature collagen decreased. The final phase of maturation occurred over the next 2 years, with grafts meeting histologic criteria of being "ligamentous" by 3 years (*Figure 2A*).

Abe *et al.* (29) performed light and electron microscopy analysis of biopsy specimens obtained from 21 knees previously reconstructed with a patellar tendon autograft. Biopsy was obtained at second look arthroscopy from 6 weeks to 15 months postoperatively. Their findings correlate with the early healing phase occurring over the first 5 months, the proliferative phase occurring from 5 to 9 months, and the maturation phase occurring beyond 9 months. They highlight in their findings that although at around 1 year postoperatively, the grafts were very similar in appearance to the native ACL on arthroscopic and light microscopic analysis, ultrastructural study suggests the grafts are still immature (*Figure 2B*).

Falconiero *et al.* (31) described similar findings in their study of hamstring (n=8) and patellar tendon (n=35) autografts. They found that collagen fiber alignment was restored around 6 months postoperatively; however, based on histologic analysis, full maturation was not achieved before 12 months postoperatively.

In their biopsy study of 37 hamstring autograft ACLRs, Sánchez *et al.* (30) observed the process of early healing occurred between 6 to 12 months postoperatively, proliferation from 13 to 18 months, and maturation from 19 to 24 months. In their study, by 24 months, specimens were histologically similar to the native ACL and characterized by the orientation of cells arranged in columns within the mature collagen matrix (*Figure 2C*).

Among the studies analyzing the ultrastructural appearance of both autograft and allograft tissue, an endpoint to the process of ligamentization has not been observed. The final ultrastructural appearance of the sampled graft tissue has commonly displayed small diameter, unimodal collagen fibril distribution, in contrast to the native ACL and hamstring tendon (HT) which consist of large diameter, bimodal collagen fibril distribution (29,32-35).

Graft healing within tunnels

Another consideration regarding graft healing is the incorporation of the graft within the bone tunnels. This differs depending on the graft type used. Bone-to-bone healing is seen in grafts that possess a bone block flanking the tendon, such as BTB grafts and quadriceps tendon (QT) grafts. Bone block healing within tunnels has been observed to resemble that of fracture healing, although somewhat more complex. Complete incorporation of the bone block in a round bone tunnel has been reported at 16 weeks postoperatively (36-38). Tendon-to-bone healing occurs through a separate process. This process is highlighted by the formation of fibrovascular tissue and Sharpey's fibers at the graft-bone interface. This fibrovascular tissue then mineralizes, followed by mineralization of the entire tendon tissue within the tunnel. The presence of Sharpey's fiber at the graft-bone interface is a marker of tendon cicatrization and has been observed beginning around 6 weeks postoperatively. Ultimately, the process of graft integration in the bone tunnel continues for 6–12 months (36,37).

Based on the results from basic science and animal studies, it is a widely held belief that bone-to-bone healing results in a quicker and more robust healing process (37,39-42). As a result of this belief, patients receiving a BTB autograft often are allowed to return to sport earlier than patients receiving a HT graft (43). However, there is a paucity of literature on rates of bone-to-bone *vs.* tendon-to-bone healing in human subjects to support this belief. To investigate this, Irvine *et al.* (44) embedded 0.8 mm tantalum beads into both HT (semitendinosus/gracilis) and BTB autografts in order to measure longitudinal motion of the grafts under various physiologic conditions using dynamic stereo X-rays. Round bone tunnels were created in both study conditions. Longitudinal motion was used a surrogate for graft healing and suspensory fixation was used in all cases. They found no difference in tibial tunnel motion or femoral tunnel motion between the HT and BTB patients at both 6 weeks and 1 year postoperatively. These results question the practice of allowing earlier return to play for patients receiving BTB grafts.

Complications

Graft failure/reinjury

A significant proportion of patients undergoing ACLR will experience a graft failure. Graft failure rates up to 40% have been reported in young, high risk patient populations (45). Failure of the ACL graft is multifactorial and can be associated with traumatic injury, incomplete or poor biologic healing, insufficient or inappropriate rehabilitation, and surgical technique (46,47). Choice of graft type has important clinical implications regarding failure risk among different patient populations.

Systematic reviews examining outcomes of adult populations undergoing ACLR have consistently found no difference in rate of failure between autograft and allograft reconstructions (48-50). However, data from the Multicenter Orthopaedic Outcomes Network (MOON), capturing

a prospective longitudinal cohort of ACLR outcomes showed that the odds of a graft rupture after an allograft reconstruction were 4 times higher compared to autograft reconstructions (51). Similarly, a recent review examining failure rates in patients under the age of 19 years of age found a significantly higher rate of failure for allografts (25.5%) compared to autograft (8.5% for BTB; 16.6% for HT), with a near 4-fold increased risk of failure in patients receiving an allograft (52).

In a systematic review and meta-analysis evaluating outcomes of QT autograft (including with and without patellar bone block) *vs.* BTB autograft and HT autograft (including semitendinosus/gracilis or semitendinosus alone, 3 or 4 strands), there was no difference in failure rate between the three autografts (53). Similarly, results from meta-analyses by Mohtadi *et al.* (54), Xie *et al.* (55), and Samuelsen *et al.* (56) showed similar rates of failure between BTB autograft and HT autograft. A systematic review from the Scandinavian Knee Ligament Registry evaluated outcomes following 45,998 ACLR and found the rate of revision was reduced by 37% with use of BTB autograft compared to HT autograft, with HT autograft resulting in a 4-fold increase in risk of revision (57). Lind *et al.* evaluated the results of 16,579 ACLR from the Danish Knee Ligament Reconstruction Registry which showed revision rates were highest in patients receiving a QT autograft (4.7%) *vs.* HT autograft (2.3%) and BTB (1.5%) autograft (58). Of the patients receiving QT autograft, 54% included a patellar bone block. When patients were stratified by age, there were increased rates of revision amongst younger, higher activity level patients, with patients 16 to 20 years of age experiencing revision rates of 10.3% for QT autograft, 4.2% for BTB autograft, and 3.8% for HT autograft.

For women 25 years of age and younger, a systematic review evaluating outcomes following ACLR found use of BTB autograft was associated with a significantly lower risk of failure compared to HT autograft (6.1% and 17.4%, respectively) (59). Similarly, amongst younger patients, both male and female, there have been consistent results showing use of HT autograft is associated with higher rates of failure compared to BTB autograft with up to 5-year follow-up data (52,60-63). A study from the MOON knee group analyzing 6-year revision rates between BTB and HT autograft reconstruction among patients 14 to 22 years of age found that high-grade preoperative knee laxity, autograft type, and age were the 3 most influential predictors of graft revision, with rates of revision being 2.1 times higher in the HT autograft group compared to the BTB group (64).

Donor site morbidity

ACLR is an overall well tolerated procedure. However, morbidity from graft harvesting when using autologous tissue is clinically relevant. According to available literature, BTB autografts incur the greatest risk of donor site morbidity when compared to HT and QT autograft (65). Harvest site morbidity seen with BTB use includes anterior knee pain, sensory loss, patella fracture, patellar tendon tear/rupture, patellar tendinopathy, quadriceps amyotrophy, and flexion contracture (66). Anterior knee pain with kneeling has been reported in up to 31% of patients receiving BTB autograft (67). In a review by Cerulli *et al.* (65), reported rates of anterior knee pain ranged from 5% to 55% for BTB autografts. The incidence of patella fracture following BTB autograft harvesting is rare, with reported rates between 0.2% to 2.3% (68,69). Similarly, patella tendon tear is infrequently seen. The results of a database of 5,364 ACLR using BTB autograft found an incidence of patella tendon injury to be 0.2% (70). One report found an incidence of extension deficit >5° of 2% for BTB autograft ACLR (71).

Anterior knee pain is also associated with HT autograft use, but at a much lower incidence (54,65,67). Other complications that are more frequently seen in HT autografts include greater residual laxity, persistent flexion contracture, and knee flexion weakness (54).

In a prospective study of 958 cases, Rousseau *et al.* reported their rate of complications following ACLR using BTB and HT autografts (7). Their results showed a significant difference in the incidence of anterior knee pain during the 2-year follow-up period for BTB and HT grafts (23.3% *vs.* 12.6%, P<0.001). BTB autograft was also associated with a significantly higher rate of contralateral ACL rupture (5% *vs.* 2%, P=0.016). Meanwhile, HT autografts were associated with a significantly higher rate of pain around the fixation material (0.8% *vs.* 13.9%, P<0.001) and graft re-rupture (3.1% *vs.* 7%, P=0.023). Other reported complications did not show a significant difference between groups and included persistent pain at 2 years (3.1% *vs.* 2.5%), extension deficit (6.6% *vs.* 10%), secondary meniscal lesion (5% *vs.* 8.3%), fracture of the patella (1.1% *vs.* 0%), and other general complications including infection and thromboembolism (7% *vs.* 4.8%). Of note, regarding patella fractures, there were only three patients that experienced a patella fracture and all were following a

BTB autograft.

Compared to BTB and HT, results following QT autograft have not been as extensively published. In a systematic review comparing results of ACLR with QT autograft *vs.* BTB and HT autograft, respectively, Mouarbes *et al.* (53) found QT autograft was associated with lower rates of anterior knee pain compared to BTB and better functional outcome scores compared to HT autograft. Other reported complications include quadriceps weakness, patella fracture (when a bone plug has been harvested), and sensory changes (72,73).

Infection

Infection following ACLR is an infrequent, yet devasting complication that can result in multiple reoperations, prolonged antibiotic therapy, graft removal and delayed revision reconstruction (74), and overall worse outcomes due to articular cartilage destruction and arthrofibrosis (75-78). The reported incidence of infection following ACLR is 0.14% to 1.7% (74,79).

It was previously believed that use of allograft tissue was associated with a higher incidence of infection, with evidence to support this (80-82). However, more recent evidence has failed to find a significant difference in infection rates between allograft and autograft reconstructions (79,80,83-85).

There have been a number of studies demonstrating use of HT autograft is associated with a higher incidence of infection compared to BTB autograft (80,83,86-88). In a large single institution cohort study, compared to BTB autograft, HT autograft and allograft were both independently associated with an increased risk of infection (odds ratio =4.39 and 5.27, respectively) (88). In another retrospective review, the use of HT autograft was associated with a 8.2 times higher risk of infection compared to BTB autograft (85). A recent meta-analysis comparing the rates of infection between BTB and HT autografts analyzed the results of 21 studies which demonstrated BTB autograft was associated with 77% lower incidence of infection compared to HT autograft. As a secondary measure, this study also found no significant difference in incidence of infection between autograft and allograft reconstructions (74).

Limited evidence is available citing the incidence of infection with QT autograft. A review of 17 studies using bone-QT autografts demonstrated a pooled incidence of infection of 1.5% (89). However, to our knowledge there currently are no existing meta-analyses comparing the incidence of infection between QT and BTB and/or HT.

Conclusions

Following reconstruction of the ACL, graft healing requires the tendon graft to undergo a continuous transformation until it is remodeled into tissue very similar to that of the native ACL, a process termed ligamentization. Evidence from human biopsy samples suggest that this process may continue for 1–3 years following ACLR.

The implication of incomplete graft healing prior to return to activity is the increased potential for graft failure. Based on prospective longitudinal cohort data, there are higher rates of graft failure following allograft reconstruction *vs.* autograft reconstruction. Amongst the autograft choices, similar rates of failure have been demonstrated for bone-patella tendon-bone, HT, and QT autografts in the general population. However, evidence from the Scandinvian and Danish registries suggest there may be higher rates of failure with QT autografts compared to bone-patella tendon-bone and HT autografts. Although similar rates of failure have been reported between bone-patella tendon-bone and HT autografts in the general population, use of HT autograft is associated with higher failure rates in younger, athletic patient populations.

Complications and infection following ACLR are infrequently seen. The most common complications include anterior knee pain, seen most frequently following BTB autograft, sensory changes, stiffness, weakness, and laxity. There is not compelling evidence to suggest allograft reconstruction carries an increased risk of infection compared to autograft reconstruction. However, there is evidence to suggest use of hamstring autograft carries a higher risk of infection compared to BTB autograft.

Acknowledgments

Funding: None.

Footnote

Peer Review File: Available at https://aoj.amegroups.com/article/view/10.21037/aoj-24-3/prf

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://aoj.amegroups.com/article/view/10.21037/aoj-24-3/coif). A.G.G. serves as the infographics editor for the *Arthroscopy* journal and serves on the editorial board. He is a member of the AOSSM Council of Delegates. He is a paid consultant with Smith

& Nephew and has received speaking fees from Ossur. The other authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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doi: 10.21037/aoj-24-3
Cite this article as: Boyd E, Endres NK, Geeslin AG. Postoperative healing and complications based on anterior cruciate ligament reconstruction graft type. Ann Joint 2024;9:30.